

=> file medline biosis biotechno

COST IN U.S. DOLLARS

SINCE FILE
ENTRY

TOTAL
SESSION

FULL ESTIMATED COST

0.15

0.15

FILE 'MEDLINE' ENTERED AT 14:03:13 ON 10 JUN 2000

FILE 'BIOSIS' ENTERED AT 14:03:13 ON 10 JUN 2000

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=> s dcr3

L1 15 DCR3

=> dup rem l1

PROCESSING COMPLETED FOR L1

L2 7 DUP REM L1 (8 DUPLICATES REMOVED)

=> d ibib abs 1-7

L2 ANSWER 1 OF 7 MEDLINE

ACCESSION NUMBER: 2000179904 MEDLINE

DOCUMENT NUMBER: 20179904

TITLE: A membrane-bound Fas decoy receptor expressed by human thymocytes.

AUTHOR: Jenkins M; Keir M; McCune J M

CORPORATE SOURCE: Gladstone Institute of Virology and Immunology, University of California, San Francisco, California 94141-9100, USA.

CONTRACT NUMBER: R01-AI40312 (NIAID)

K08-AI01425 (NIAID)

SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (2000 Mar 17) 275 (11) 7988-93.

Journal code: HIV. ISSN: 0021-9258.

PUB. COUNTRY: United States

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals; Cancer Journals

ENTRY MONTH: 200006

ENTRY WEEK: 20000603

AB Human thymocytes at several stages of maturation express Fas, yet resist apoptosis induction through its ligation. A proximal step in apoptotic signaling through Fas is implicated in this resistance, as these cells undergo normal levels of apoptosis induction after exposure to tumor necrosis factor-alpha. We studied the Fas receptors expressed in human thymocytes to search for mechanisms of receptor-mediated inhibition of

Fas signaling in these cells. We describe here a unique, membrane-bound form of Fas receptor that contained a complete extracellular domain of Fas but that lacked a death domain due to alternative splicing of exon 7. This

Fas decoy receptor (FDR) was shown to have nearly wild-type ability to bind native human Fas ligand and was expressed predominantly at the plasma membrane. Unlike soluble forms of Fas receptor, FDR dominantly inhibited apoptosis induction by Fas ligand in transfected human embryonic kidney cells. Titration of FDR in Fas-expressing cells suggests that FDR may operate through the formation of mixed receptor complexes. FDR also dominantly inhibited Fas-induced apoptosis in Jurkat T cells. In mixing

experiments with wild-type Fas, FDR was capable of inhibiting death signaling at molar ratios less than 0.5, and this relative level of FDR:wild type message was observed in at least some thymocytes tested.

The

data suggest that Fas signal pathways in primary human cells may be regulated by expression of a membrane-bound decoy receptor, analogous to the regulation of tumor necrosis factor-related apoptosis inducing ligand (TRAIL)-induced apoptosis by decoy receptors.

L2 ANSWER 2 OF 7 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2000122600 MEDLINE
DOCUMENT NUMBER: 20122600
TITLE: Overexpression of M68/DcR3 in human
gastrointestinal tract tumors independent of gene
amplification and its location in a four-gene cluster.
AUTHOR: Bai C; Connolly B; Metzker M L; Hilliard C A; Liu X;
Sandig
CORPORATE SOURCE: V; Soderman A; Galloway S M; Liu Q; Austin C P; Caskey C T
Department of Human Genetics, Merck Research Laboratories,
West Point, PA 19486-0004, USA.. chang_bai@merck.com
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE
UNITED STATES OF AMERICA, (2000 Feb 1) 97 (3) 1230-5.
Journal code: PV3. ISSN: 0027-8424.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
OTHER SOURCE: GENBANK-AF217793; GENBANK-AF217794; GENBANK-AF217795;
GENBANK-AF217796
ENTRY MONTH: 200005
ENTRY WEEK: 20000501

AB Fas-mediated apoptosis is an important regulator of cell survival, and abnormalities in this system have been shown to result in a number of human pathological conditions. A secreted member of the tumor necrosis factor receptor superfamily, DcR3, was recently reported to be amplified in human lung and colon cancers as a negative regulator of Fas-mediated apoptosis. We identified this gene, which we call M68. M68 genomic DNA, mRNA, and protein levels were examined in a series of human gastrointestinal tract tumors. Using M68 immunohistochemistry and a scoring system similar to that used for HER-2/neu, we found that M68 protein was overexpressed in 30 of 68 (44%) human adenocarcinomas of the esophagus, stomach, colon, and rectum. Tumors examined by Northern blot revealed M68 mRNA highly elevated in a similar fraction of primary tumors from the same gastrointestinal tract regions, as well as in the colon adenocarcinoma cell lines SW480 and SW1116. Further, we found M68 protein to be overexpressed in a substantial number of tumors in which gene amplification could not be detected by fluorescence in situ hybridization or quantitative genomic PCR, suggesting that overexpression of M68 may precede amplification in tumors. Finally, we find that M68 lies within a four-gene cluster that includes a novel helicase-like gene (NHL) related to RAD3/ERCC2, a plasma membrane Ras-related GTPase and a member of the stathmin family, amplification or overexpression of which may also contribute to cell growth and tumor progression.

L2 ANSWER 3 OF 7 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2000098473 MEDLINE
DOCUMENT NUMBER: 20098473
TITLE: Over-expression of the decoy receptor 3 (DcR3)
gene in peripheral blood mononuclear cells (PBMC) derived
from silicosis patients.
AUTHOR: Otsuki T; Tomokuni A; Sakaguchi H; Aikoh T; Matsuki T;
Isozaki Y; Hyodoh F; Ueki H; Kusaka M; Kita S; Ueki A
CORPORATE SOURCE: Department of Hygiene, Kawasaki Medical School, Kurashiki,
Okayama, Japan.. takemi@med.kawasaki-m.ac.jp
SOURCE: CLINICAL AND EXPERIMENTAL IMMUNOLOGY, (2000 Feb) 119 (2)
323-7.
Journal code: DD7. ISSN: 0009-9104.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 200005
ENTRY WEEK: 20000501

AB Dysregulation of apoptosis, particularly in the Fas/Fas ligand (FasL) pathway, is considered to be involved in the pathogenesis of autoimmune diseases such as systemic lupus erythematosus (SLE). Recently, a soluble decoy receptor, termed decoy receptor 3 (Dcr3), that binds FasL and inhibits FasL-induced apoptosis, has been identified. Silicosis is clinically characterized not only by respiratory disorders but by immunological abnormalities. We have found that serum soluble Fas (sFas) levels are elevated in silicosis patients and that sFas message is dominantly expressed in PBMC derived from these patients. This study examined Dcr3 gene expression in PBMC derived from patients with silicosis, SLE, or progressive systemic sclerosis (PSS), and compared it with that in healthy volunteers (HV). The relative expression level of

the Dcr3 gene was examined in PBMC derived from 37 patients with silicosis without clinical symptoms of autoimmune disease, nine patients with SLE, 12 patients with PSS, and 28 HV using the semiquantitative multiplex-reverse transcriptase-polymerase chain reaction (MP-RT-PCR).

The correlation between the relative expression level of the Dcr3 gene and multiple clinical parameters for respiratory disorders and immunological abnormalities in individuals with silicosis was analysed. The Dcr3 gene was significantly over-expressed in cases of silicosis or SLE when compared with HV. In addition, the Dcr3 relative expression level was positively correlated with the serum sFas level in silicosis patients. It is unclear, however, whether over-expression of the Dcr3 gene in silicosis is caused by chronic silica exposure, merely accompanies the alteration in Fas-related molecules, or precedes the clinical onset of autoimmune abnormalities. It will be necessary to study these patients further, establish an in vitro model of human T cells exposed recurrently to silica compounds, and resolve whether the increase in Dcr3 mRNA expression is a cause or consequence of disease.

L2 ANSWER 4 OF 7 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 1999253915 MEDLINE
DOCUMENT NUMBER: 99253915
TITLE: A newly identified member of tumor necrosis factor receptor superfamily (TR6) suppresses LIGHT-mediated apoptosis.
AUTHOR: Yu K Y; Kwon B; Ni J; Zhai Y; Ebner R; Kwon B S
CORPORATE SOURCE: Department of Microbiology and Immunology and Walther Oncology Center, Indiana University School of Medicine and the Walther Cancer Institute, Indianapolis, Indiana 46202, USA.
CONTRACT NUMBER: AI28125 (NIAID)
DE12156 (NIDCR)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 May 14) 274 (20) 13733-6.
Journal code: HIV. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
OTHER SOURCE: GENBANK-AF134240
ENTRY MONTH: 199908

AB TR6 (decoy receptor 3 (Dcr3)) is a new member of the tumor necrosis factor receptor (TNFR) family. TR6 mRNA is expressed in lung tissues and colon adenocarcinoma, SW480. In addition, the expression of TR6 mRNA was shown in the endothelial cell line and induced by phorbol 12-myristate 13-acetate/ionomycin in Jurkat T leukemia cells. The open reading frame of TR6 encodes 300 amino acids with a 29-residue signal sequence but no transmembrane region. Using histidine-tagged recombinant TR6, we screened soluble forms of TNF-ligand proteins with immunoprecipitation. Here, we demonstrate that TR6 specifically binds two cellular ligands, LIGHT (herpes virus entry mediator (HVEM)-L) and Fas

ligand (FasL/CD95). These bindings were confirmed with HEK 293 EBNA cells transfected with LIGHT cDNA by flow cytometry. TR6 inhibited LIGHT-induced cytotoxicity in HT29 cells. It has been shown that LIGHT triggers apoptosis of various tumor cells including HT29 cells that express both lymphotoxin beta receptor (LTbetaR) and HVEM/TR2 receptors. Our data suggest that TR6 inhibits the interactions of LIGHT with HVEM/TR2 and LTbetaR, thereby suppressing LIGHT-mediated HT29 cell death. Thus, TR6 may play a regulatory role for suppressing in FasL- and LIGHT-mediated cell death.

L2 ANSWER 5 OF 7 MEDLINE
ACCESSION NUMBER: 1999227321 MEDLINE
DOCUMENT NUMBER: 99227321
TITLE: Apoptosis control by death and decoy receptors.
AUTHOR: Ashkenazi A; Dixit V M
CORPORATE SOURCE: Department of Molecular Oncology, Genentech Inc, 1 DNA Way,
South San Francisco, CA 94080, USA.
SOURCE: CURRENT OPINION IN CELL BIOLOGY, (1999 Apr) 11 (2) 255-60.
Ref: 66
Journal code: AOE. ISSN: 0955-0674.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199907
ENTRY WEEK: 19990705
AB The death receptors Fas and tumor necrosis factor receptor 1 (TNFR1) trigger apoptosis upon engagement by their cognate death ligands. Recently, researchers have discovered several novel homologues of Fas and TNFR1: DR 3, 4, 5, and 6 function as death receptors that signal apoptosis, whereas DcR 1, 2, and 3 act as decoys that compete with specific death receptors for ligand binding. Further, mouse gene knockout studies have enabled researchers to delineate some of the signaling pathways that connect death receptors to the cell's apoptotic machinery.

L2 ANSWER 6 OF 7 MEDLINE
ACCESSION NUMBER: 1999087312 MEDLINE
DOCUMENT NUMBER: 99087312
TITLE: Apoptosis. Death deceiver [news; comment].
COMMENT: Comment on: Nature 1998 Dec 17;396(6712):679-703
AUTHOR: Green D R
SOURCE: NATURE, (1998 Dec 17) 396 (6712) 629-30.
Journal code: NSC. ISSN: 0028-0836.
PUB. COUNTRY: ENGLAND: United Kingdom
Commentary
News Announcement
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199903
ENTRY WEEK: 19990304

L2 ANSWER 7 OF 7 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 1999087326 MEDLINE
DOCUMENT NUMBER: 99087326
TITLE: Genomic amplification of a decoy receptor for Fas ligand in lung and colon cancer.
AUTHOR: Pitti R M; Marsters S A; Lawrence D A; Roy M; Kischkel F C;
Dowd P; Huang A; Donahue C J; Sherwood S W; Baldwin D T; Godowski P J; Wood W I; Gurney A L; Hillan K J; Cohen R L; Goddard A D; Botstein D; Ashkenazi A
CORPORATE SOURCE: Department of Molecular Oncology, Molecular Biology, and Immunology, Genentech, Inc., South San Francisco,

California 94080, USA.
.SOURCE: NATURE, (1998 Dec 17) 396 (6712) 69-73.
Journal code: NSC. ISSN: 0028-0836.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
OTHER SOURCE: GENBANK-AF104419
ENTRY MONTH: 199903

AB Fas ligand (FasL) is produced by activated T cells and natural killer cells and it induces apoptosis (programmed cell death) in target cells through the death receptor Fas/Apo1/CD95. One important role of FasL and Fas is to mediate immune-cytotoxic killing of cells that are potentially harmful to the organism, such as virus-infected or tumour cells. Here we report the discovery of a soluble decoy receptor, termed decoy receptor 3 (DcR3), that binds to FasL and inhibits FasL-induced apoptosis. The DcR3 gene was amplified in about half of 35 primary lung and colon tumours studied, and DcR3 messenger RNA was expressed in malignant tissue. Thus, certain tumours may escape FasL-dependent immune-cytotoxic attack by expressing a decoy receptor that blocks FasL.

=> s tnfr 6 alpha

L3 0 TNFR 6 ALPHA

=> s tnfr6 alpha

L4 0 TNFR6 ALPHA

=> tnfr6

TNFR6 IS NOT A RECOGNIZED COMMAND

The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s tnfr6

L5 0 TNFR6

=> s tnfr

L6 1429 TNFR

=> tnfr.6

TNFR.6 IS NOT A RECOGNIZED COMMAND

The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s tnfr.6

L7 0 TNFR.6

=> d his

(FILE 'HOME' ENTERED AT 14:02:32 ON 10 JUN 2000)

FILE 'MEDLINE, BIOSIS, BIOTECHNO' ENTERED AT 14:03:13 ON 10 JUN 2000

L1 15 S DCR3
L2 7 DUP REM L1 (8 DUPLICATES REMOVED)
L3 0 S TNFR 6 ALPHA
L4 0 S TNFR6 ALPHA
L5 0 S TNFR6
L6 1429 S TNFR
L7 0 S TNFR.6

.=> s decoy receptor

L8 169 DECOY RECEPTOR

=> s l8 and fas

L9 17 L8 AND FAS

=> dup rem l9

PROCESSING COMPLETED FOR L9

L10 9 DUP REM L9 (8 DUPLICATES REMOVED)

=> s l10 not l2

L11 5 L10 NOT L2

=> d ibib abs 1-5

L11 ANSWER 1 OF 5 MEDLINE

ACCESSION NUMBER: 2000200790 MEDLINE

DOCUMENT NUMBER: 20200790

TITLE: Lipopolysaccharide induces expression of APO2 ligand/TRAIL in human monocytes and macrophages.

AUTHOR: Halaas O; Vik R; Ashkenazi A; Espevik T

CORPORATE SOURCE: Institute of Cancer Research and Molecular Biology, The Norwegian University of Science and Technology (NTNU), N-7489 Trondheim, Norway.

SOURCE: SCANDINAVIAN JOURNAL OF IMMUNOLOGY, (2000 Mar) 51 (3) 244-50.

Journal code: UCW. ISSN: 0300-9475.

PUB. COUNTRY: ENGLAND: United Kingdom

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals; Cancer Journals

ENTRY MONTH: 200006

ENTRY WEEK: 20000602

AB Monocytes express cytotoxic factors of the tumour necrosis factor (TNF) ligand superfamily, including TNF and **Fas** ligand, both on the cell surface and in secreted form. In this report, we show that human monocytes and monocyte-derived macrophages stimulated with lipopolysaccharide (LPS) express APO2 ligand (APO2L, TRAIL), a recently discovered cytotoxic member of the TNF ligand superfamily. LPS increased the transcription of APO2L mRNA in monocytes and macrophages. Flow cytometric analysis showed low surface and high intracellular levels of APO2L, and LPS increased the expression of both. In addition, LPS increased the monocyte- and macrophage-mediated cytotoxicity against the APO2L-sensitive Jurkat and RPMI-8226 cells. Addition of the APO2L-binding **decoy receptor** 1 (DcR1)-Fc fusion protein inhibited the cytotoxicity by 30-70%. LPS also stimulated the release of soluble APO2L from the monocytes and macrophages. Monocytic phagocytosis of target cells

was increased by LPS and partially inhibited by DcR1-Fc. Taken together, these data demonstrate a novel mechanism of cytotoxicity mediated by LPS-activated human monocytes and macrophages.

L11 ANSWER 2 OF 5 MEDLINE

ACCESSION NUMBER: 2000016594 MEDLINE

DOCUMENT NUMBER: 20016594

TITLE: Control of apoptosis signaling by Apo2 ligand.

AUTHOR: Marsters S A; Pitti R A; Sheridan J P; Ashkenazi A

CORPORATE SOURCE: Department of Molecular Oncology, Genentech, Inc, South San

Francisco, California 94080, USA.

SOURCE: RECENT PROGRESS IN HORMONE RESEARCH, (1999) 54 225-34.

Ref: 26

Journal code: R1D. ISSN: 0079-9963.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)

LANGUAGE: English
ENTRY MONTH: 200002
ENTRY WEEK: 20000204

AB Apo2 ligand (Apo2L, also called TRAIL) is a member of the tumor necrosis factor (TNF) cytokine family. The closest homolog of Apo2L is CD95 (Fas/Apo1) ligand, to which it has 24% amino acid sequence identity. Similar to CD95L, Apo2L activates rapid apoptosis in many types of cancer cells; however, whereas CD95L mRNA expression is restricted mainly to activated T cells, natural killer cells, and immune-privileged sites, Apo2L mRNA occurs in a wide variety of tissues. Most normal cells appear to be resistant to Apo2L's cytotoxic action, suggesting the existence of mechanisms that can protect against apoptosis induction by Apo2L. The first receptor described for Apo2L, called death receptor 4 (DR4), contains a cytoplasmic "death domain"; DR4 transmits the apoptosis signal carried by Apo2L. We have identified three additional receptors that bind to Apo2L. One receptor, called DR5, contains a cytoplasmic

death domain and signals apoptosis much like DR4. The DR4 and DR5 mRNAs are expressed in many normal tissues and tumor cell lines. The second receptor, designated **decoy receptor 1** (DcR1), is a phospholipid-anchored cell-surface protein that lacks a cytoplasmic tail. The third receptor, called DcR2, is structurally similar to DR4 and DR5 but has a truncated cytoplasmic death domain and does not transmit a

death signal. The mRNAs for DcR1 and DcR2 are expressed in multiple normal tissues but in few tumor cell lines. Transfection experiments indicate that DcR1 and DcR2 act as decoys that prevent Apo2L from inducing apoptosis through DR4 and DR5. These decoy receptors thus represent a novel mechanism for regulating sensitivity to a pro-apoptotic cytokine directly at the cell's surface. The preferential expression of these inhibitory receptors in normal tissues suggests that Apo2L may be useful as an anticancer agent that induces apoptosis in cancer cells while sparing normal cells.

L11 ANSWER 3 OF 5 BIOSIS COPYRIGHT 2000 BIOSIS
ACCESSION NUMBER: 1999:339262 BIOSIS
DOCUMENT NUMBER: PREV199900339262
TITLE: Apoptosis regulation by death and decoy receptors.
AUTHOR(S): Ashkenazi, Avi (1)
CORPORATE SOURCE: (1) Department of Molecular Oncology, Genentech Inc., 1 DNA

Way, South San Francisco, CA, 94080 USA
SOURCE: FASEB Journal, (April 23, 1999) Vol. 13, No. 7, pp. A1336.

Meeting Info.: Annual Meeting of the American Societies for

Experimental Biology on Biochemistry and Molecular Biology
99 San Francisco, California, USA May 16-20, 1999 American Societies for Experimental Biology
. ISSN: 0892-6638.

DOCUMENT TYPE: Conference
LANGUAGE: English

L11 ANSWER 4 OF 5 BIOSIS COPYRIGHT 2000 BIOSIS
ACCESSION NUMBER: 1999:325367 BIOSIS
DOCUMENT NUMBER: PREV199900325367
TITLE: Apoptosis control by death and decoy receptors.
AUTHOR(S): Ashkenazi, Avi; Dixit, Vishva M.
CORPORATE SOURCE: Dep. Mol. Oncol., Genentech Inc., 1 DNA Way, South San Francisco, CA 94080 USA
SOURCE: Current Opinion in Cell Biology, (April, 1999) Vol. 11, No.

2, pp. 255-260.
ISSN: 0955-0674.

DOCUMENT TYPE: General Review

LANGUAGE: English

L11 ANSWER 5 OF 5 BIOSIS COPYRIGHT 2000 BIOSIS
ACCESSION NUMBER: 1998:274245 BIOSIS
DOCUMENT NUMBER: PREV199800274245
TITLE: APO2 ligand: A novel lethal weapon against malignant glioma.
AUTHOR(S): Rieger, Johannes; Naumann, Ulrike; Glaser, Tamara; Ashkenazi, Avi; Weller, Michael (1)
CORPORATE SOURCE: (1) Lab. Med. Neuro-Oncol., Dep. Neurol., Univ. Tuebingen, Sch. Med., Hoppe-Seyler-Str. 3, 72076 Tuebingen Germany
SOURCE: FEBS Letters, (May 1, 1998) Vol. 427, No. 1, pp. 124-128. ISSN: 0014-5793.
DOCUMENT TYPE: Article
LANGUAGE: English

AB APO2L (TRAIL) is a novel CD95L (**Fas**/APO-1-L) homologous cytotoxic cytokine that interacts with various receptors which transmit (DR4, DR5) or inhibit (DcR1, DcR2) an apoptotic signal. Here, we report that human glioma cell lines preferentially express mRNAs for agonistic death receptors DR4 (8/12) and DR5 (11/12) rather than the death-inhibitory decoy receptors DcR1 (4/12) and DcR2 (2/12). Ten of 12 cell lines are susceptible to APO2L-induced apoptosis. The resistant cell lines, U138MG and U373MG, are cross-resistant to CD95L-induced apoptosis.
Similar to CD95L-induced apoptosis, APO2L-induced apoptosis is inhibited by ectopic expression of the caspase inhibitor, crm-A, or of bcl-2, or by coexposure to the corticosteroid, dexamethasone, or the lipoxygenase inhibitor, nordihydroguarectic acid. There is no correlation between p53 genetic status of the cell lines and their susceptibility to APO2L-induced apoptosis, but the latter is moderately enhanced by ectopic expression of wild-type p53. APO2L targeting may be a promising approach for selectively targeting apoptosis to human malignant glioma cells.

=> s (fas/apo-1)

'APO-1' IS NOT A VALID FIELD CODE
'APO-1' IS NOT A VALID FIELD CODE
'APO-1' IS NOT A VALID FIELD CODE
L12 0 (FAS/APO-1)

=> s apo-1

L13 2228 APO-1

=> and decoy receptor

AND IS NOT A RECOGNIZED COMMAND

The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter "HELP COMMANDS" at an arrow prompt (=>).

=> s l13 and decoy receptor

L14 1 L13 AND DECOY RECEPTOR

=> d ibib abs

L14 ANSWER 1 OF 1 BIOSIS COPYRIGHT 2000 BIOSIS
ACCESSION NUMBER: 1998:274245 BIOSIS
DOCUMENT NUMBER: PREV199800274245
TITLE: APO2 ligand: A novel lethal weapon against malignant glioma.
AUTHOR(S): Rieger, Johannes; Naumann, Ulrike; Glaser, Tamara; Ashkenazi, Avi; Weller, Michael (1)
CORPORATE SOURCE: (1) Lab. Med. Neuro-Oncol., Dep. Neurol., Univ. Tuebingen,

SOURCE: Soc. Med., Hoppe-Seyler-Str. 3, 72074 Tuebingen Germany
FEBS Letters, (May 1, 1998) Vol. 427 No. 1, pp. 124-128.
ISSN: 0014-5793.

DOCUMENT TYPE: Article

LANGUAGE: English

AB APO2L (TRAIL) is a novel CD95L (Fas/APO-1-L) homologous cytotoxic cytokine that interacts with various receptors which transmit (DR4, DR5) or inhibit (DcR1, DcR2) an apoptotic signal. Here, we report that human glioma cell lines preferentially express mRNAs for agonistic death receptors DR4 (8/12) and DR5 (11/12) rather than the death-inhibitory decoy receptors DcR1 (4/12) and DcR2 (2/12). Ten of 12 cell lines are susceptible to APO2L-induced apoptosis. The resistant cell lines, U138MG and U373MG, are cross-resistant to CD95L-induced

apoptosis.

Similar to CD95L-induced apoptosis, APO2L-induced apoptosis is inhibited by ectopic expression of the caspase inhibitor, crm-A, or of bcl-2, or by coexposure to the corticosteroid, dexamethasone, or the lipoxygenase inhibitor, nordihydroguaretic acid. There is no correlation between p53 genetic status of the cell lines and their susceptibility to

APO2L-induced

apoptosis, but the latter is moderately enhanced by ectopic expression of wild-type p53. APO2L targeting may be a promising approach for

selectively

targeting apoptosis to human malignant glioma cells.

=> s tumor necrosis factor receptor

L15 4834 TUMOR NECROSIS FACTOR RECEPTOR

=> s tumor necrosis factor receptor 6

L16 0 TUMOR NECROSIS FACTOR RECEPTOR 6

=> log y